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Case Report
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An obstructive retrosternal thyroid mass mimicking asthma

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ABSTRACT

The term retrosternal/intrathoracic goiter was first defined by Haller in 1749. It is usually located in the anterior mediastinum. When it reaches large sizes, it can cause various pressure symptoms. Respiratory distress may occur due to tracheal compression, difficulty swallowing due to esophageal compression, and venous fullness findings may occur as a result of pressure on vascular structures. Although rare, it can be life-threatening; it may also cause respiratory failure, which may develop as a result of sudden growth that may occur secondary to intrathyroidal hemorrhage or cystic degeneration. In this case, we aimed to present a patient who experienced respiratory distress due to a thyroid mass extending retrosternally and causing tracheal compression, and whose symptoms persisted despite optimal asthma treatment.

Keywords: Asthma, retrosternal thyroid mass, plonjon goitre

INTRODUCTION

Asthma is a chronic inflammatory disease affecting more than 300 million people worldwide. While most asthma patients respond well to current treatments, approximately 5-10% of asthma patients do not respond adequately to treatment despite optimum treatment. When evaluating patients with symptoms suggestive of difficult asthma, the differential diagnosis of difficult asthma should be made first and conditions mimicking asthma should be revealed, then comorbidities and conditions that make asthma control difficult, triggering factors should be identified and controlled, then the drug compliance of the patients should be evaluated and finally the most appropriate treatment option should be applied for the patients, taking into account the asthma phenotypes.

The term retrosternal/intrathoracic goiter was first defined by Haller in 1749.[1] It is usually located in the anterior mediastinum. When it reaches large sizes, it can cause various pressure symptoms.[2] Respiratory distress may occur due to tracheal compression, difficulty swallowing due to esophageal compression, and venous fullness findings may occur as a result of pressure on vascular structures. Although rare, it can be life-threatening; It may also cause respiratory failure, which may develop as a result of sudden growth that may occur secondary to intrathyroidal hemorrhage or cystic degeneration. In this case, we aimed to present a patient who experienced respiratory distress due to a thyroid mass extending retrosternally and causing tracheal compression, and whose complaints did not go away despite optimum asthma treatment.

CASE

A 76-year-old female patient, who had been receiving asthma treatment for 20 years, applied to our outpatient clinic with the complaint of increased shortness of breath.

In her detailed anamnesis, it was learned that she had a known history of hypertension, vertigo, osteoporosis and a previous cerebral transient ischemic attack. The patient, who is a housewife, has not worked in any job before. It was learned that the patient was sensitive to odors and did not use cigarettes or alcohol. The patient was receiving inhaled corticosteroid, long-acting beta agonist, leukotriene receptor antagonist and short acting beta agonist treatment for asthma, but her symptoms still continued. There was no other respiratory symptoms like stridor and wheezing and also there was no esophageal compression symptoms like dysphagia, odynophagia, regurgitation or chest pain. There was no involuntary weight loss or night sweats.

On physical examination, breathing sounds were evaluated as rough. There were no rales or rhonchi. There was no use of accessory respiratory muscles or cyanosis. The saturation value was measured as 96% with a fingertip pulse oximeter.

Expansion of the upper mediastinum was observed on chest radiography. The trachea was observed deviated to the left (Figure 1). A restrictive and obstructive pattern was observed in the pulmonary function test (PFT). FEV1: 0.8 (47%) FVC:1.31 (59%) FEV1/FVC:61% was measured (Figure 2).

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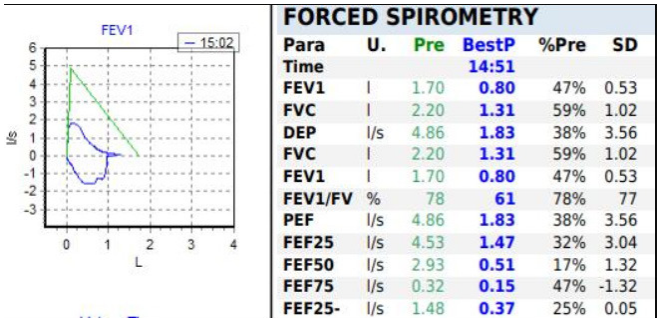
Figure 1. The pulmonary function test showed a fixed obstruction



FORCED SPIROMETRY

Para	U.	Pre	BestP	%Pre	SD
Time			14:51		
FEV1	l	1.70	0.80	47%	0.53
FVC	l	2.20	1.31	59%	1.02
DEP	l/s	4.86	1.83	38%	3.56
FVC	l	2.20	1.31	59%	1.02
FEV1	l	1.70	0.80	47%	0.53
FEV1/FV	%	78	61	78%	77
PEF	l/s	4.86	1.83	38%	3.56
FEF25	l/s	4.53	1.47	32%	3.04
FEF50	l/s	2.93	0.51	17%	1.32
FEF75	l/s	0.32	0.15	47%	-1.32
FEF25-	l/s	1.48	0.37	25%	0.05

Figure 2. The PTS's showed a fixed obstruction

Contrast-enhanced thorax computerized tomografy was planned due to upper mediastinum widening detected on chest radiography. In the thorax CT; it was reported that the size of the right lobe of the thyroid gland had increased significantly, and coarse calcifications and hypodense nodular appearances were observed in the right lobe of the thyroid gland. The lower contours of the right lobe of the thyroid extend to the neighborhood of the arcus aorta and occasionally contact the main vascular structures in the upper mediastinal area, and it was observed that the trachea secondary to retrosternal goiter was deviated to the left and narrowed the tracheal lumen. Additionally, a few pure calcific subpleural nodules measuring 13x6 mm in size were also seen in the anterior upper lobe of the right lung (**Figure 3**).

Figure 3. Chest computed tomography revealed a giant mass lesion pressured on the trachea from the right side

PET-CT was planned due to the suspicion of malignancy of the nodular lesions. In the PET-CT report, a hypodense mass lesion of 8 cm in size was seen in the right lobe of the thyroid gland, extending to the retrosternal area and creating significant pressure on the trachea from the right side, narrowing the airway significantly.

It was thought that the patient's respiratory symptoms unresponsive to bronchodilator treatment were the result of the retrosternally extending giant thyroid mass compressing the trachea, and the patient was referred to general surgery for surgical treatment. The patient did not come for follow-up later.

DISCUSSION

Asymptomatic patients with retrosternal goiter account for about 15-30% of all such patients.[3] Retrosternal goiter should be considered in cases such as tracheal deviation and mediastinal expansion, which are detected incidentally on a chest radiograph taken for any reason in asymptomatic patients.[4,5] In cases with retrosternal goiter, it usually occurs due to pressure on vital organs between the sternum and vertebral column.

While retrosternal goiter causes stridor, shortness of breath or cough due to the compression of the trachea, in some cases, difficulty in swallowing occurs due to esophageal compression. In our case, there was a complaint of shortness of breath despite the use of inhaler therapy, and there were no pathological findings on physical examination. In cases like ours, alternative diagnoses should be considered in situations where there is no response to optimum treatment. Hoarseness due to recurrent laryngeal nerve compression or invasion, or rarely, vena cava superior (VCS) syndrome, pulmonary hypertension or ischemic attacks due to superior vena cava, pulmonary artery or carotid artery compression.

By looking at the flow-volume curve, it can be interpreted whether the lesion is intrathoracic or extrathoracic. In fixed obstruction, the airway diameter does not change throughout the respiratory cycle. The clinical symptoms and changes in PFT of fixed type of obstruction in the upper respiratory tract and large airways are well known.

During the evaluation of our patient, who was diagnosed with asthma and applied to the outpatient clinic with complaints of shortness of breath, goiter causing tracheal stenosis was detected in the chest X-ray and thorax CT. PFT showed fixed obstruction. The patient was referred to surgery.

As a result, monitoring fixed obstruction in PFT in patients presenting with shortness of breath should be a warning about possible upper airway stenosis.

CONCLUSION

Upper airway stenosis is one of the pathologies that should be considered in patients with severe and progressive shortness of breath during follow-up. Plungant goiter is one of the causes of obstruction, and both intrathoracic and extrathoracic PFT findings can be detected depending on the goiter compression. In conclusion, imaging and surgical evaluation are crucial for early diagnosis and intervention, as they significantly improve quality of life and survival. Therefore, as in our case a multidisciplinary approach is essential.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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